Complete Thrombosis of a Giant Distal Middle Cerebral Artery Aneurysm

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Summary

Complete thrombosis of a giant cerebral aneurysm is rarely reported and peripheral giant middle cerebral artery (MCA) aneurysms are similarly rare. We present a case of complete spontaneous thrombosis of a giant sylvian artery aneurysm that had previously been wrapped at surgery.

Introduction

Giant MCA aneurysms usually arise at the bifurcation of the MCA. Giant distal MCA aneurysms are rarely reported [1,2,3]. Partial thrombosis within giant aneurysms is common but the factors that determine progression to complete thrombosis are poorly understood [1]. Partial thrombosis is not thought to reduce the risk of subarachnoid haemorrhage from a giant aneurysm but may result in embolic phenomena and occasionally progression to complete thrombosis [1,2,3]. The long-term stability of spontaneously thrombosed giant aneurysms is uncertain [4].

Case Report

A thirty-six year-old woman with a history of migraine presented in May 1990 with pain in the left temporal region, right sided sensory disturbance and weakness involving the right hand. No abnormality was detected on examination and her symptoms were attributed to hyperventilation. The symptoms progressed over three months with sensory disturbance involving the right arm and face, difficulty swallowing and right limb weakness. On examination in September 1990 weakness and hypereflexia was noted in the right leg. Cerebral angiography demonstrated a bi-lobed partially thrombosed aneurysm arising from the left MCA beyond the bifurcation (no images available).

At surgery one lobe of the aneurysm was thrombosed. The aneurysm neck was exposed and the M2 segment was dissected back to the bifurcation. It was not possible to position a clip satisfactorily because of calcification at the aneurysm neck and the aneurysm was therefore wrapped with isobutyl-cyanoacrylate. The patient recovered from surgery without any deterioration and remained clinically stable. Embolisation was attempted in 1992 but was abandoned due to arterial spasm. The patient was therefore managed conservatively. The angiographic projections at the time of attempted embolisation in 1992 demonstrated the aneurysm with sylvian branches arising distally from both locules (figure 1A,B).

The patient's symptoms returned in 1997 when she developed a speech disorder and partial seizures which occurred with increasing frequency as many as ten times per month. The

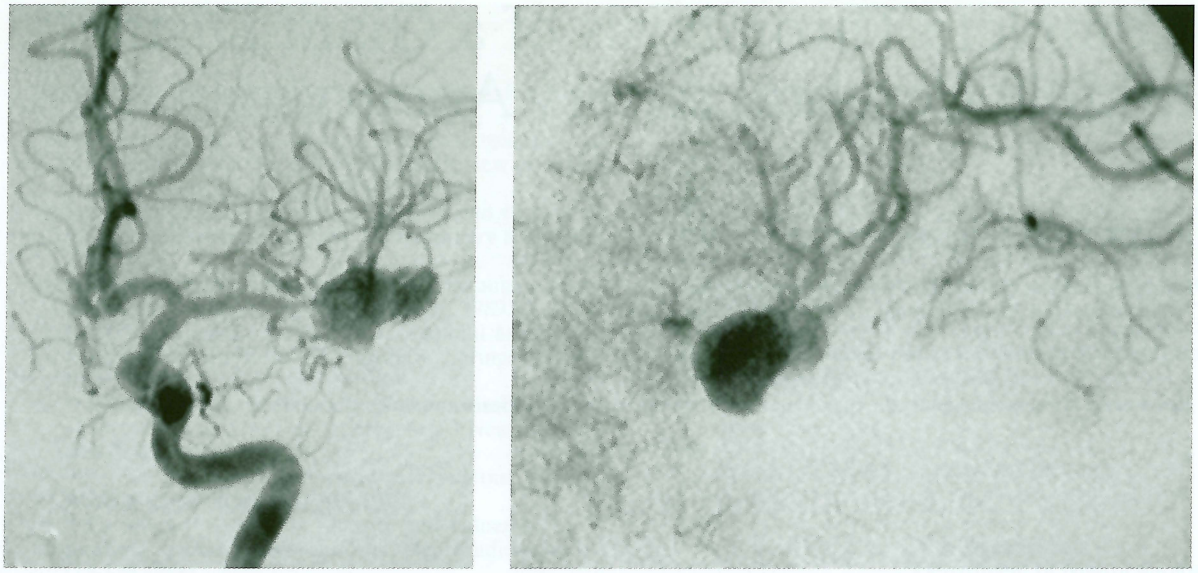

Figure 1 A) Left internal carotid view demonstrating the distal location of the left MCA aneurysm. B) Capillary phase showing sylvian branches arising distally from the aneurysm.

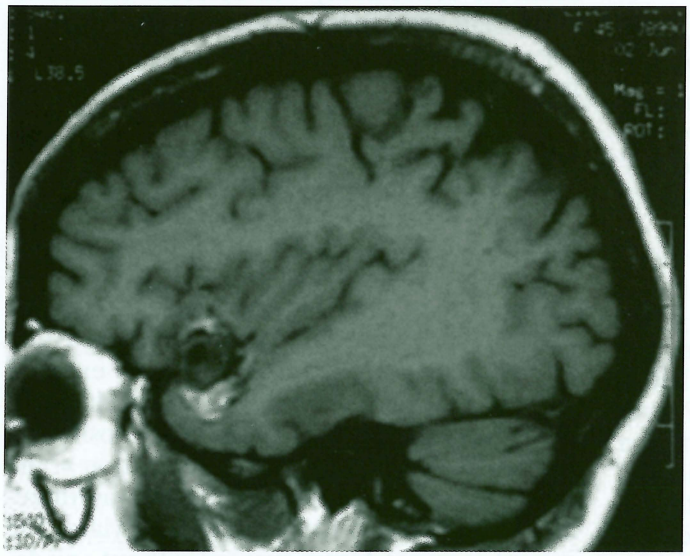

Figure 2 Coronal T1-weighted image showing the thrombosed sylvian MCA aneurysm.

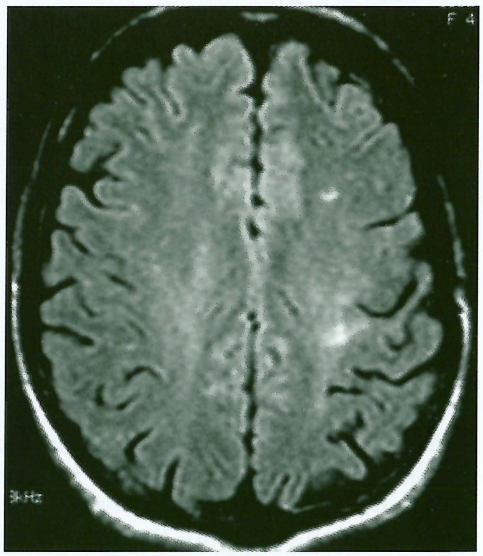

Figure 3 Axial FLAIR image showing ischaemic lesions in the left hemisphere.

seizures responded to medication but in 1999 the patient developed right leg weakness lasting a few hours.

MRI in 1999 showed that the giant left sylvian aneurysm was largely thrombosed (figure 2). Several small ischaemic lesions were noted in the left hemisphere (figure 3) and in view of the patient's continued symptoms (which were attributed to both local mass effect and cerebral emboli from the aneurysm) the patient was referred for consideration of embolisation of the left sylvian aneurysm. Cerebral angiography demonstrated spontaneous thrombosis of the aneurysm and its parent vessel (figure 4).

Discussion

Giant intracranial aneurysms occur most frequently at the carotid bifurcation (59%) and basilar tip. The MCA bifurcation and anterior communicating artery are also common sites for giant aneurysms [5]. Up to 13% of MCA aneurysms are giant and the majority of giant MCA aneurysms occur at the bifurcation [2]. The origin of a giant aneurysm from the M2 segment beyond the bifurcation in this case is rare. Peripheral cerebral aneurysms are uncommon accounting for 2% to 9% of middle cerebral artery aneurysms and a similar proportion of anterior cerebral artery aneurysms [6].

The distal location of the giant aneurysm and its non-saccular morphology in this case raise the possibility of various aetiologies including: mycotic aneurysm, atherosclerotic aneurysm, dissecting aneurysm, or serpentine type aneurysm [5]. The classification of non-saccular distal aneurysms remains controversial and many descriptive terms are used to describe overlapping phenomena.

Without histology, the classification of the distal non-saccular aneurysm in this case is speculative. There was no evidence of an infective aetiology and the presentation and natural history of the aneurysm did not favour an acute dissection. The presentation with symptoms of mass affect, the findings at surgery (calcification) and the clinical progress of the aneurysm suggest an atheromatous aneurysm or a dolichoectatic type giant aneurysm as classified by Mizutani [7]. The aneurysm is unlikely to be atheromatous because of the patient's age at presentation and the aneurysm location. The dolichoectatic type dissecting aneurysms described in Mizutani's paper involved the basilar artery, were fusiform and tortuous and contained thrombus. There was no evidence of atheromatous change in the aneurysms in his series. It was proposed that dolichoectatic type aneurysms grew by a process of repeated intramural haemorrhage and six of the eight aneurysms in his series grew to a giant size during the follow-up period. All of the patients presented with local symptoms due to mass effect from the aneurysm and three died following rupture of the aneurysm. Histology in four of the cases demonstrated laminar thrombus within the lumen of the aneurysm, fragmentation of the internal elastic lamina and multiple dissection of the thickened intima.

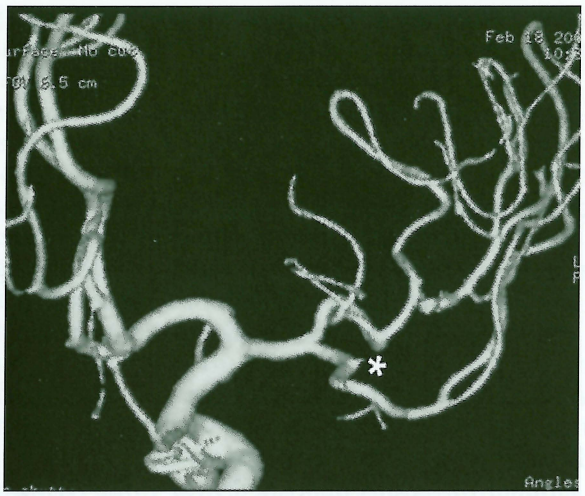

Figure 4 3-D reconstruction demonstrating occlusion of the M2 segment (*) and aneurysm.

Similar histological findings have been described in younger patients presenting with fusiform aneurysms involving the vertebrobasilar artery and the petrous, cavernous and intradural segments of the internal carotid artery 5. In older patients fusiform aneurysms in these locations are typically atherosclerotic and are associated with hypertension, aortic aneurysms and positive syphilis serology [5].

The aneurysm in our case originated on the distal M2 segment and extended to involve the proximal M3 segments. While the distal MCA is not a traditional location for a dolichoectatic type aneurysm the clinical presentation with symptoms related to mass effect and emboli, the presence of thrombus, and the aneurysm morphology with involvement of both proximal M3 segments raise the possibility of a fusiform type aneurysm.

Although the patient's symptoms in this case were not suggestive of an acute dissection it is possible that a dissection of the M2 and M3 segments that did not extend subadventitially and rupture may have been the primary event. Our patient had a history of migraine that preceded her presentation (rather than becoming a feature subsequent to her presentation) and cerebral artery spasm and dissection are well recognised in association with migraine al-

though this usually involves the internal carotid artery or vertebrobasilar system [8]. Failure to remodel and propagation of the thrombus following acute dissection in this instance could result in a similar lesion to the dolichoectatic type aneurysm proposed. The possibility that aneurysms classified as distinct histological entities by Mitzutani could have similar or identical angiographic features (and aetiologies) but could represent opposite ends of a pathological continuum was one of the significant criticisms of his paper.

The onionskin appearance of a giant aneurysm is a characteristic finding on MRI and is thought to correspond to layers of intraluminal thrombus deposited over time. Distinguishing intraluminal from intramural thrombus within a thrombosed aneurysm is not possible without histological examination. The capacity to distinguishing a giant thrombosed aneurysms from a dolichoectatic type or chronic dissecting type aneurysms is similarly restricted on the basis of imaging data. The term giant aneurysm however is limited as it fails to convey any information about the lesion other than an agreed minimum size.

The presentation in this case was similar to giant MCA aneurysms previously reported in the literature. Five of the twelve partially thrombosed aneurysms in Whittle's series were giant MCA aneurysms. The following historical and presenting features were noted in these five patients: headache (n=2), temporal lobe seizures (n=2), progressive right brachial paresis and syncope [1]. Two of the five partially thrombosed MCA aneurysm presented with subarachnoid haemorrhage. Pain referred to the eye and ear has also been reported in a patient with a thrombosed MCA aneurysm [3,5]. The pathogenesis of the referred pain was not discussed but it is likely to be related to dural irritation by the aneurysm.

Embolic ischaemic events have been attributed to both unruptured and ruptured aneurysms of all sizes [5]. Qureshi reported a 3.3% incidence of strokes or TIAs from the aneurysm sac in 269 patients with unruptured aneurysms [9]. Of the nine patients identified four had MCA aneurysms and three had internal carotid aneurysms. In a review of the literature a further forty-one patients presenting with stroke or TIA were identified. Thirty-seven percent of these patients had MCA aneurysms. In all cases cardiac and cervical carotid disease had been excluded. The most common presentation was with TIAs, which could be associated with relatively small aneurysms without evidence of luminal thrombus. While the rate of recurrent TIAs was lower in patients that underwent surgery the benefit was obscured by postoperative complications. The long-term risk of major disabling stroke was felt to be low with either conservative or surgical management [9].

Medical treatment with antiplatelet agents was proposed to reduce platelet aggregation within the aneurysm sac in patients presenting with TIAs. The theoretical disadvantage of impairing platelet mediated fibroblast migration and scar formation within the wall of the aneurysm potentially inhibiting the healing process was discussed however the authors concluded that in patients with small (<10 mm) aneurysm aspirin may be used to reduce the risk of TIAs [9].

Intra-aneurysmal thrombus occurs in approximately 50% of giant aneurysms however the factors that determine the balance between thrombogenesis and thrombolysis within giant aneurysms are poorly understood [1]. Previous studies have suggested that the size of the aneurysm is important in determining the risk of in-situ thrombosis [1,2,9]. Factors that are thought to influence the balance between thrombus formation and degradation include: the ratio of aneurysm volume to orifice size, endothelial injury due to turbulent blood flow, distortion of the parent artery, subarachnoid haemorrhage, vasospasm, prothrombotic states and antifibrinolytic agents [1,2,9]. These and other factors that influence aneurysm formation and growth such as genetic susceptibility, somatic mutations, growth factor disorders, abnormal vascular remodelling and arterial segmental vulnerability vary between and within individuals [10].

Complete thrombosis of intracranial aneurysms is uncommon and occurs most frequently following subarachnoid haemorrhage and in fusiform or giant saccular aneurysms. Complete aneurysm thrombosis following subarachnoid haemorrhage occurs in 1-2% of cases and has been reported in up to 55% of giant aneurysms demonstrated on CT scans [11]. Spontaneous thrombosis of the majority of a giant aneurysm has been reported to occur as quickly as one month following diagnosis of the lesion [3].

Parent or distal vessel thrombosis has been reported in association with aneurysmal thrombosis following subarachnoid haemorrhage, during treatment with antifibrinolytic agents, spontaneously in giant aneurysms, and following surgical and endovascular procedures[5,11]. In cases of giant aneurysm thrombosis where the parent vessel occludes simultaneously the patient may present with an intracranial mass and a "false negative" angiogram. The mixed signal mass on MRI imaging may be mistaken for a cavernoma or haemorrhagic tumour[1,3]. In our case angiography demonstrated a small stump at the MCA trifurcation demonstrating the location of the occluded vessel.

The timing of complete thrombosis is unknown in this case. Attempted embolisation in 1992 two years following surgery demonstrated a bilobed partially thrombosed aneurysm and it is possible that the thrombosed lobe noted at surgery had recanalised at this stage. It is also unlikely that the subsequent aneurysmal thrombosis was directly related to surgery.

MRI in 1999 showed virtually complete thrombosis of the aneurysm but as the patient remained symptomatic she was referred for repeat angiography and consideration of embolisation. Angiography demonstrated complete occlusion of the aneurysm and proximal M2 branch.

The long-term stability of thrombosed aneurysms is uncertain and recanalisation of thrombosed aneurysms is well recognised both in the acute setting of angiogram negative subarachnoid haemorrhage and in cases of spontaneously thrombosed aneurysms without subarachnoid haemorrhage[4].

In Atkinson's case of a thrombosed P1/P2 segment aneurysm the parent vessel remained patent but MRI three months after the ictus demonstrated recanalisation of the aneurysm, which was subsequently treated surgically.

Growth of completely thrombosed giant aneurysms is also recognised due to continued intramural haemorrhage from vasa vasorum[5].

There is no published literature to guide further management in our case.

As recanalisation and subsequent haemorrhage remain a possibility the case will be followed-up by MRI examinations. MRI one year following angiography confirmed complete occlusion of the aneurysm and M2 segment.

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